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Maggie Ladd  
*Iowa State University*

Eric Reinertson  
*Iowa State University*

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# Medical Management and Hoof Care of Acute Laminitis in the Horse

Maggie Ladd, DVM<sup>†</sup>  
Eric Reinertson, DVM, MS<sup>††</sup>

Laminitis is a costly and frustrating disease for the horse owner and veterinarian alike. It is difficult to treat successfully and retain the horse's previous level of performance and soundness. Regardless of the cause of laminitis, the pathophysiology is still unclear. Early recognition, identification, and prompt aggressive treatment of horses that may suffer from laminitis as a secondary effect of a systemic disease, however, is the key to successful recovery. This article will review the definition, suspected pathophysiology, clinical signs, and diagnosis of laminitis, as well as current medical recommendations and hoof care considerations in the laminitic horse.

## Brief Review

Laminitis is described by *Adams' Lameness in Horses* as inflammation of the laminae of the hoof.<sup>1</sup> There are two types or classes of laminitis: 1) the physical tearing of laminae due to severe concussive forces, typical of road founder, and 2) physiological, such as that which is secondary to a systemic metabolic disease or septicemia. This article focuses on physiological laminitis.

To better understand the pathophysiology of laminitis, it is necessary to first review the anatomy of the hoof. Interdigital laminae suspend the distal phalanx (P3) from the hoof capsule. The interdigital laminae are made up of the dermal laminae, which arise from the corium and dermis of the skin. Their base adheres to P3. They contain blood vessels that nourish both the dermal and epidermal laminae.<sup>1</sup> There are arteriovenous (AV) anastomoses at the base

of each lamina.<sup>2</sup> The epidermal laminae are an extension of the cornified epidermis and deeper layers. They intermesh with the dermal laminae to connect the hoof wall to P3. The epidermal laminae receive blood via diffusion from the vessels in the dermal laminae.<sup>1</sup>

Physiological laminitis is a peripheral vascular disease of the hoof that is a manifestation of systemic metabolic diseases such as colitis, colic, endometritis, carbohydrate overload, or respiratory disease. It can also be incited by hormonal fluctuations and drugs, such as corticosteroids.<sup>3</sup> Rotation of P3 due to laminitis is caused by a combination of the laminae dying and releasing their hold and the deep digital flexor tendon pulling P3 away from the hoof wall. Epidermal laminae are affected first in acute laminitis because of their more distant blood supply.<sup>1</sup> Congestion and edema of the dermal laminae cause epidermal laminar necrosis at the dermal junction. When the necrosis extends to the dermal laminae and the deep digital flexor pulls P3, rotation results.<sup>1</sup>

## Pathophysiology

There are several proposed causes of the laminar necrosis seen in the hoof during acute laminitis. These include vasoconstriction within the hoof, microthrombosis, perivascular edema, and arteriovenous (AV) shunting.<sup>1,3</sup> Nuclear scintigraphy studies of carbohydrate overload-induced laminitis have shown an increased blood flow to the hoof along with AV shunting.<sup>4</sup> Other kinds of laminitis, such as that induced by ingestion of black walnuts, have shown an initial decrease in blood flow followed by an increase later in the course of the disease.<sup>3</sup> Either vasoconstriction of the capillary bed or increased blood flow and AV shunting cause the laminae to suffer from ischemic injury,<sup>1,3,5</sup> and edema and pain result. Pain causes a cat-

<sup>†</sup>Maggie Ladd is a 1997 graduate of the Iowa State University College of Veterinary Medicine.

<sup>††</sup>Dr. Eric Reinertson is an Associate Professor in Veterinary Clinical Sciences at the Iowa State University College of Veterinary Medicine.



Eric Reinertson, ISU

This is the outward appearance of a hoof with chronic laminitis. PIII has rotated and the horse is unable to work due to this condition.



Eric Reinertson, ISU

Radiographic changes with laminitis will usually occur 48 hours after the onset of clinical signs. In this radiograph, note the rotation of PIII and the areas of activity in the bone.

echolamine release, which leads to further vasoconstriction and more ischemic injury.

A coagulopathy has been noted in endotoxin-induced laminitis. Thrombus formation, as demonstrated histologically,<sup>6</sup> leads to endothelial damage and capillary blockage, thus compounding the ischemia caused by hemodynamic changes. Alterations have also been noted in the intrinsic coagulation system during the developmental stages of acute endotoxin-induced laminitis. These changes could cause inappropriate clotting or thrombus formation.<sup>1</sup>

### Clinical Signs and Diagnosis

The diagnosis of laminitis is made from the clinical signs. It can affect either the front feet or all four feet. When the front feet are affected, the horse rocks its weight back on its hind feet by placing them well up under itself. Affected horses are reluctant to move. If all four feet are involved, the horse will spend much of its time lying down. The hooves may feel warm to hot, and there are increased digital pulses. Acutely laminitic horses will show signs of intense pain, indi-

cated by increased heart rate, respiratory rate, and sometimes temperature. They may also have injected mucous membranes. Tenderness over the toe or the entire sole region of the hoof in response to hoof testers is also a sign.<sup>1,5</sup>

Radiographic changes will usually occur 48 hours after the onset of clinical signs.<sup>3</sup> It is important to take baseline radiographs to monitor the progress of the disease and amount of PIII rotation. It has been suggested that horses with PIII rotation of <5.5 degrees have a favorable prognosis for return to athletic performance whereas horses with a PIII rotation of 6.8-11.5 degrees have a guarded prognosis. Horses whose PIII has rotated more than 11.5 degrees have a very poor prognosis.<sup>7</sup> Abrupt change or extreme rotation in any case greatly decreases the final prognosis and may indicate failure of medical management.

### Medical Therapy

Acute laminitis is a medical emergency.<sup>1</sup> Clinical exhibition of laminitis may not appear until 12-18 hours after the inciting

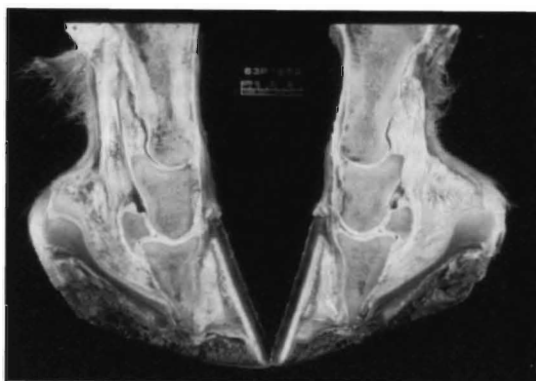
cause, thus it is important to recognize conditions that may result in laminitis and treat the horse to prevent laminitis from occurring.<sup>1,3</sup> Medical therapy is empirical and retrospective, because there is no proof of which vascular events are occurring in the hoof during an acute attack of laminitis.<sup>3</sup>

The goals of medical therapy are: 1) institute therapy before the horse actually becomes lame; 2) Minimize or neutralize the insult (for example, with colitis, minimize the intestinal absorption of toxins and limit the amount of thrombi that form in the microvasculature); 3) reduce the pain and prevent or lessen the pain/hypertension cycle that develops; 4) reduce or prevent permanent laminar damage; 5) improve digital hemodynamics; and 6) prevent further movement of PIII.<sup>3</sup>

There are three basic classes of drugs used in the treatment of laminitis to achieve the above goals. The nonsteroidal anti-inflammatory drugs (NSAIDs), vasodilators,<sup>8</sup> and antithrombotics<sup>9</sup> are all used because they have been purported to help laminitic horses in the past.<sup>3</sup> Other drugs that have been used with variable efficacy include anti-endotoxins,<sup>3</sup> and pentoxifylline.<sup>10,11</sup>

Commonly used NSAIDs are phenylbutazone, flunixin meglumine, ketoprofen, and DMSO.<sup>3</sup> Of these drugs, phenylbutazone is the cheapest and most often used to decrease the inflammation, edema, and pain within the hoof. Baxter<sup>3</sup> suggests a dose of 4.4 mg/kg every twelve hours for three to four days, then decreasing to 2.2 mg/kg every twelve hours for seven to ten days. Phenylbutazone acts by inhibiting cyclooxygenase, thereby decreasing prostaglandin synthesis and preventing vasoconstriction.<sup>12</sup> It also decreases platelet aggregation and has anticoagulant activity which prevents formation of thrombi in the microvasculature of the hoof.<sup>3</sup> Phenylbutazone has potent analgesic properties which help to break the pain/hypertension/reflex vasoconstriction cycle that develops in acute laminitis.<sup>1</sup>

Phenylbutazone can be used alone, or in combination with flunixin meglumine (1.1 mg/kg BID)<sup>3</sup> if there is a septicemic process involved. Flunixin meglumine is a potent analgesic, anti-inflammatory, and antipyretic NSAID that may help improve blood flow in the hoof through its inhibition of pros-



Yoshiya Niyo, ISU

**A post-mortem cross-section through a hoof shows the deviation of PIII due to laminitis. PIII has actually perforated through the hoof wall in this horse.**

taglandin synthesis.<sup>3</sup> Flunixin meglumine may also be used alone for the treatment of the clinical signs of laminitis.

Ketoprofen is another NSAID that has been used in the treatment of laminitis.<sup>12,13</sup> It is a propionic acid derivative that inhibits arachidonic acid and prostaglandin production, as well as acting on the lipooxygenase system.<sup>12</sup> In general, it has fewer side effects and is safer to use in horses; however, a higher than labeled dose is needed to achieve clinical response in acute laminitis cases.<sup>13</sup> In addition, it is the most expensive of the NSAIDs, and therefore is cost-prohibitive for long term therapy.<sup>3</sup>

Finally, DMSO has been used to prevent reperfusion injury to the ischemic tissues in the hoof. This is based only on clinical response to treatment, as there is no proof of the occurrence of reperfusion injury.<sup>3</sup> The recommended dosage is 0.1-1 g/kg IV BID. This can be mixed in a 5% electrolyte solution wherein 250 ml of a 90% dextrose solution is added to 3 liters of fluids.<sup>3</sup> It is important to avoid concentrations of greater than 20% to prevent hemolysis of red blood cells. During intravenous DMSO administration, the horse should be monitored daily for signs of hemolysis.

Anti-thrombotics are used because it has been documented with histopathology that microthrombi have formed in laminitic hooves.<sup>2,6</sup> The use of anti-thrombotics is purely preventative and will not change an existing thrombotic condition. Commonly used anti-thrombotics include aspirin at 10-20 mg/kg every other day and heparin at 40-100 IU/kg IV or subcutaneously TID or QID.<sup>3</sup> Aspirin inhibits platelet aggregation and

thromboxane synthesis thereby decreasing the formation of thrombi.<sup>12</sup> The recommended dose is low enough to be used with NSAIDs and has no toxic side effects.

Heparin is a cofactor to antithrombin III which inhibits thrombin and down-regulates prothrombin.<sup>9,14</sup> There is some controversy about the efficacy of heparin in treating laminitis. In carbohydrate-induced laminitis, heparin seems to make a difference, but in cases in which the horse is already ill with gastrointestinal disease before initiation of heparin therapy, there does not seem to be much response.<sup>14</sup> The level of antithrombin III in horses correlates with how well they respond to heparin therapy.<sup>9,14</sup> Horses with normal levels of antithrombin III respond better than horses with lower levels of antithrombin III. Heparin therapy has been shown to reduce the incidence of laminitis from 90% to 20% with carbohydrate-induced laminitis, especially if heparin therapy was begun before the horse actually had clinical signs of laminitis and presumably before the antithrombin III levels had decreased.<sup>1,9,14</sup> It is important when using heparin to monitor activated partial thromboplastin values daily.

Vasodilators are considered effective because of suspected vasoconstriction and ischemia occurring in the hoof. They act to promote digital circulation and dilation of microvascular beds. This class includes perineural anesthesia, acetylpromazine, phenoxybenzamine, isoxsuprine, nitric oxide/*l*-arginine, and glyceryl trinitrate.

Perineural anesthesia acts to decrease pain-related peripheral vasoconstriction.<sup>1</sup> A disadvantage to using local anesthesia is that it could encourage the horse to move around more and cause more physical trauma to the weakened laminae.

Acetylpromazine is an adrenergic blocking drug that may act to decrease systemic hypertension, rather than increase digital perfusion.<sup>3,12</sup> Because of the decrease in systemic hypertension, there is less pressure on the capillary bed of the hoof and less shunting of blood in the hoof. In addition, there are fewer catecholamines released, and therefore less reflex vasoconstriction.<sup>3</sup> Acepromazine has also been shown to increase hoof perfusion in the healthy standing horse, but the perfusion does not reach

the dorsal laminae.<sup>3,15</sup>

Phenoxybenzamine is an alpha-adrenergic non-competitive blocker that has been shown to increase cutaneous blood flow. It acts in a similar manner to acetylpromazine.

Isoxsuprine is better for long-term use in chronic laminitis horses. It causes vascular smooth muscle relaxation, and at high doses, can decrease blood viscosity and platelet aggregation, which may be helpful in acute cases of laminitis. It has minimal systemic vasodilation effects in a normal horse.<sup>3,15</sup>

Nitric oxide has been used transdermally with mixed responses.<sup>8</sup> Nitric oxide synthase (NOS) acts on its substrate *l*-arginine to relax vascular smooth muscle and cause vasodilation.<sup>8</sup> Experimentally, IV infusion of *l*-arginine has caused immediate reperfusion of laminar tissues in an acute laminitis case with little effect on the systemic blood pressure.<sup>8</sup>

Topical glyceryl trinitrate has reduced the bounding pulses and lameness associated with laminitis.<sup>8</sup> Side effects of topical glyceryl trinitrate are irritation to the skin where it is applied and lowered systemic blood pressure.<sup>8</sup> Topical glyceryl trinitrate seems to work in some horses but not in all of them.

Anti-endotoxin therapy has had questionable efficacy in cases of laminitis.<sup>3</sup> Its efficacy is questionable, and at this time, its only value would be to reduce the systemic effects of endotoxins in endotoxin-induced laminitis concurrent with colitis, endometritis, or pleuropneumonia.

Pentoxifylline is a relatively new drug that is on the market for treatment of endotoxemia in the horse. It functions to increase RBC filterability and decrease sedimentation rate of erythrocytes.<sup>10</sup> It has also been shown to improve microcirculation.<sup>10</sup> Endotoxin causes a significant increase in blood levels of Tumor Necrosis Factor (TNF), Interleukin6 (IL6), Prostaglandin F2 $\alpha$  (PGF2 $\alpha$ ), and Thromboxane B2 (TBB2). *In vitro* studies with pentoxifylline indicate that in combination with endotoxin, it reduces the amount of TNF, IL6, and TBB2 in the blood.<sup>10</sup> It also has been shown to improve regional blood flow, vasodilate, and prevent platelet aggregation.<sup>10,11</sup> Its direct effect in laminitis cases has yet to be determined. It may help

improve digital circulation and prevent thrombi formation in the hoof in cases of endotoxin-induced laminitis; however, several studies suggest that it may take several days of therapy to have a clinical effect.<sup>10,11,14</sup>

### Hoof Care

Hoof care in an acute case of laminitis focuses on preventing or halting the movement of PIII away from the hoof wall and providing relief from pain. It can involve anything from simply putting the horse in a deep, soft sand stall to trimming flares and long hoof walls, using wedge shoes or pads, and providing frog support.

The best initial hoof care is to place the horse in a soft sand stall or one that is deeply bedded.<sup>1</sup> This allows the horse to bury its toes in the sand, thus relieving the pull of the deep digital flexor tendon on PIII, which can cause further damage to the dorsal laminae.

Horses with excessively long toes should have the toes trimmed short and squared off to ease break-over and lessen the pull of the deep digital flexor tendon on PIII as the horse moves. This will also help lessen the shearing forces that are put on the lamina by long toes and flares in the hoof wall.<sup>3,16</sup>

Heel elevation is also important in cases of acute laminitis. This can be achieved with an 18 degree heel wedge pad or elevated heel shoe that can be either glued or taped in place. This will theoretically decrease the pull of the deep digital flexor tendon on PIII, thus lessening the stress put on the injured laminae. This is especially important with cases of contralateral limb lameness.

Frog support can help prevent rotation of PIII. This can be achieved through the use of lily pad shoes that are taped on the hoof, or simply with a roll of gauze taped over the caudal one-third of the frog.<sup>16</sup> It is important not to place anything over the dorsal part of the frog because doing so could occlude the blood vessels in the frog that supply the hoof, causing ischemic injury and sloughing of the hoof.

Other treatments include surgical deep digital flexor tenectomy. This is not recommended, except as a salvage procedure.<sup>16</sup> Other corrective shoeing should be left until

the pain and inflammation in the hoof have subsided.<sup>16</sup> Dorsal hoof wall resection is not recommended except in cases where there is clear radiographic evidence of separation of the hoof capsule from PIII.<sup>1,3,16</sup>

### Summary

Acute laminitis is an emergency that needs to be treated promptly to avoid or lessen the amount of permanent damage that occurs. It is important to initiate treatment as soon as possible after the inciting cause of laminitis, even before clinical signs of laminitis appear. By doing this, one can prevent or minimize damage that occurs in the hoof. Useful medical therapeutics include NSAIDs such as phenylbutazone and DMSO, anti-thrombotics such as heparin or aspirin, and vasodilators like acetylpromazine or glyceryl trinitrate. It may also be useful to try newer drugs such as pentoxifylline, especially if therapy is being instituted before the actual onset of clinical signs of laminitis. It is also important to remember to institute preventive hoof care in cases of laminitis. This includes putting the horse in a deeply bedded or sand stall, trimming long flares from its hooves, providing adequate frog support, and elevating its heels, especially if there is a lameness in the contralateral limb. Early recognition of diseases that may cause laminitis and aggressive preventive therapy will help the horse retain or return to its usual activity.♦

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## What's Your Radiographic Diagnosis?

*Elizabeth A. Riedesel, DVM, DACVR<sup>†</sup>*

A 3 year-old, neutered male, Bassett Hound presented for evaluation of acute onset of vomiting which had progressed to include anorexia and weight loss over a one week period. The dog had initially responded to symptomatic therapy. However, at home he continued to refuse food and had resumed vomiting. The description of the dog's "bringing up of food" was that of a passive action with no retching or abdominal contraction. Physical examination revealed normal body temperature, pulse rate, and respiratory rate. The dog was bright and alert but was not very active. No abdominal abnormalities were noted upon palpation. Thoracic auscultation was normal.

The history suggested regurgitation prompting differential diagnoses of esophageal disease. A barium swallow was performed and no esophageal motility or obstructive abnormalities were identified. However, an area of irregularity of the esophageal mucosa was identified. Endoscopic assessment was planned. Blood work revealed a moderate stress leukogram and normal liver and renal function.

<sup>†</sup>Dr. Elizabeth Riedesel is Associate Professor in Veterinary Clinical Sciences at the Iowa State University College of Veterinary Medicine. She is Section Leader of Radiology in the Veterinary Teaching Hospital.

On the following day, repeat abdominal palpation was performed and a firm mass was palpated in the cranial ventral abdomen. The dog had been observed to retch and vomit the prior evening. Prior to endoscopy, abdominal radiographs were taken. See Figure 1.

### Radiographic Findings

Barium is present in the stomach and cranial small intestinal tract persistent from the previous esophageal evaluation. The barium in the intestine is diluted in opacity. A rough edged, 3 cm diameter circular radiolucent filling defect is seen in the pyloric antrum on the right lateral view. This filling defect is not seen on the VD view. The duodenum and small intestinal loops that are filled with barium and gas are mildly to moderately dilated. The area of greatest dilation ends with a rounded area of irregular opacity in the right caudal abdomen at the plane of L5-7. The rounded area contains a mixture of short linear to elliptical gas mixed with barium and a very thin linear 1.5 cm radiopaque line. Several focal narrowings of the gastric lumen and affected small intestine indicate the presence of peristalsis.

**Please turn to page 96 for the diagnosis.**